Relation between admission time, haemodynamic measurements, and prognosis in acute myocardial infarction*

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SUMMARY Pulmonary arterial end-diastolic pressure, cardiac index, and stroke work index were measured via a thermistor-tipped balloon catheter and monitored for 51 ± 51 hours in 226 patients admitted with an acute myocardial infarction (184 survivors and 42 non-survivors). Mortality was related to time of admission after onset of symptoms of infarction. Of 69 patients in group A 13 died in hospital (18·8%) one to four hours after onset; in group B (five to eight hours after onset) eight of 71 patients (11%) died five to eight hours after onset; four of 26 patients in group C (15%) died nine to 12 hours after onset; 15 of 42 patients (36%) in group D died 13 to 24 hours after onset; and two of 18 patients in group E died (11%) more than 24 hours after onset. Irrespective of admission time, haemodynamic findings in survivors were significantly better than in non-survivors. During the first eight to 12 hours after onset of infarction cardiac index and stroke work index were normal or above normal, with raised left ventricular filling pressures. In patients admitted later, this compensatory mechanism had often collapsed. Where pump failure with subnormal cardiac index and stroke work index were present mortality was increased. All four patients dying from acute myocardial rupture had significantly higher values of cardiac index and stroke work index and lower values of pulmonary artery end-diastolic pressure compared with those dying from other causes.

Although the initial haemodynamic values give some prognostic information, longitudinal analysis provides insight into the evolving myocardial disturbance and compensatory mechanisms. If the initial values of pulmonary artery end-diastolic pressure and cardiac and stroke work indices remain normal or become stable after a transient disturbance in the acute phase, prognosis is good. If, however, these values deteriorate or remain abnormal, prognosis is poor. Typically such patients have suffered large infarctions with a tendency to expansion. If the haemodynamic situation during the first 24 hours after onset of infarction remains stable for 12 to 15 hours, haemodynamic monitoring may be stopped; the chance of relapse in such patients was found to be below 10%. Late deterioration, usually manifest by further pain or by electrocardiographic or enzyme changes, should be an indication to restart haemodynamic monitoring so that treatment can be chosen and adjusted optimally. These haemodynamic measurements in patients treated traditionally with vasodilators, positive inotropic agents, and fluid will form the basis for comparison of measurements in patients who are now treated within the first eight hours with selective intracoronary thrombolysis and, if possible, with adjacent intracoronary balloon dilatation of the underlying coronary artery stenosis.

The introduction of the double-lumen Swan-Ganz catheter[1] has increased our knowledge of the haemodynamic changes in acute myocardial infarction[2–7] and the necessary treatment resulting from them. The initial measurements of pulmonary arterial end-diastolic pressure and cardiac output, and derivation of cardiac stroke work index are already of clinical and prognostic importance.[8–12] Haemodynamic findings

in the early phase of myocardial infarction, however, are subject to continuous change because of alterations in myocardial wall stiffness, influence of circulating endogeneous catecholamines, possible extension and or expansion of the infarction, and local compensatory changes by the healthy parts of the myocardium.[12-14] These regulatory and adaptive changes within the first 24 hours have not always been taken into consideration. The timing of the measurements in relation to the onset of infarction and comparison with the preceding and succeeding findings are of considerable importance with respect to the clinical assessment and prognosis.

In this study the relation between the time and individual values of haemodynamic measurements, as well as their apparent prognostic significance, have been analysed in 226 patients who came to the coronary care unit in the early phase of an acute myocardial infarction.

Subjects and methods

Two hundred and twenty-six patients (172 men, 54 women) with an acute myocardial infarction were studied haemodynamically immediately after admission to hospital. After detailed explanation about the nature of the haemodynamic measurements, the advantages with respect to better clinical management, and mention of the possible complications, all patients gave their consent. Transmural inferior wall infarction occurred in 120 patients (in-hospital mortality 12%) and transmural anterior wall infarction in 98 patients (in-hospital mortality 28%); eight patients had a non-transmural infarction (in-hospital mortality 13%). A total of 42 patients (18·6%) died during the hospital phase. The mean age of the survivors was 58·8±10·6 years and that of the non-survivors 67·6±9·9 years (standard deviation).

The time of onset of the infarction was determined as precisely as possible. Patients were subdivided into groups according to the time between the presumed moment of onset of infarction and the first haemodynamic measurements. In 69 patients (30·5%) the interval was one to four hours (time group A), in 71 patients (31%) five to eight hours (time group B), in 26 patients (11%) nine to 12 hours (time group C), in 42 patients (18·6%) 13 to 24 hours (time group D), and in 18 patients (8%) more than 24 hours (time group E). As a rule, within the first hour after admission a double-lumen Swan-Ganz catheter was passed transcutaneously via an anticubital vein into the pulmonary artery, with the aid of fluoroscopy.

Initially the pressures in the right atrium, right ventricle, pulmonary artery, and pulmonary wedge position were recorded. Pulmonary arterial pressure was then monitored continuously. The systemic

arterial pressure was taken by cuff, except in shocked patients where it was obtained via a Teflon cannula in the brachial or femoral artery. The cardiac output and stroke volume were determined by the thermodilution method using a bedside cardiac output computer.* The cardiac index (CI) and stroke volume index (SVI) were then determined and stroke work index (SWI) calculated by the formula:

SWI [g m/m²] = [SVI 0·66 (mean systolic arterial pressure) − PAEDP] × 0·0136.

Table 1 *Limiting values for separating patients into different haemodynamic groups*

Group	Clinical picture	CI (l/min per m²)	PAEDP (mmHg)
I	(a) Normal haemodynamics	>2·2−3·5	<18
	(b) Hyperkinesis	≥3·5	<18
II	Isolated pulmonary congestion	>2·2	≥18
III	Isolated hypoperfusion	≤2·2	<18
IV	Pulmonary congestion + hypoperfusion	≤2·2	≥18

The patients were divided into four haemodynamic groups according to the values of the pulmonary artery end-diastolic pressure and cardiac index (Table 1). A separate analysis was also undertaken by dividing the patients into four groups according to stroke work index values (above 60 g m/m², 41 to 60 g m/m², 21 to 40 g m/m² and 20 g m/m² or less). The mean duration of measurements was 51±51 hours (standard deviation). All causes of death during the hospital phase were recorded.

Results

Initially we found normal haemodynamic values in 85 patients (group Ia), irrespective of the admission time (Fig. 1, Table 2). Their prognosis was good (mortality rate 9·4%). In a further 30 patients (13·3%) there was a hyperdynamic situation with a cardiac index of more than 3·5 l/min per m² (group Ib) and none died. Prognosis with a mortality rate of about 20% was

Table 2 *Initial haemodynamic findings in 226 patients with acute myocardial infarction*

Haemo-dynamic group	No. of patients	%	Mortality No.	%	CI (l/min per m²)	PAEDP (mmHg)
Ia	85	37·6	8	9·4	2·8±0·4	12·3±2·8
Ib	30	13·3	0	0	4·2±0·5	12·6±2·8
II	67	29·6	15	22·43	1±0·7	23·1±5·3
III	24	10·6	5	20·8	1·9±0·3	12·3±3·5
IV	20	8·9	14	70·0	1·8±0·4	24·8±6·3

Note: Cardiac index (CI) and pulmonary arterial end-diastolic pressure (PAEDP) results are group means ± standard deviation.
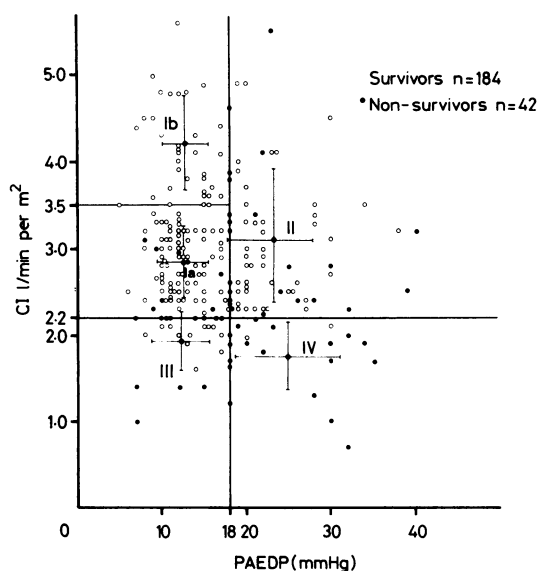
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Fig. 1 *Initial haemodynamic findings in 226 patients with acute myocardial infarction. Cardiac index (CI) is plotted against pulmonary arterial end-diastolic pressure (PAEDP). Group Ia = normal haemodynamics, group Ib = hyperkinesis, group II = isolated pulmonary congestion, group III = isolated hypoperfusion, group IV = pulmonary congestion + hypoperfusion. (See Table 2.)*

nearly equal in cases of isolated pulmonary congestion (group II) and isolated low output (group III). If both pulmonary congestion and low output were present (group IV), the prognosis was drastically worsened (mortality rate 70%). The mean cardiac index of the survivors was $3 \cdot 0 \pm 0 \cdot 77$ l/min per m^2 (SD) and that of the non-survivors $2 \cdot 3 \pm 0 \cdot 89$ l/min per m^2; their pulmonary end-diastolic pressure was $16 \cdot 0 \pm 6 \cdot 3$ mmHg and $21 \cdot 5 \pm 8 \cdot 7$ mmHg, respectively.

The isolated analysis of stroke work index (Fig. 2, Table 3) also provided important prognostic information, which became even more precise by also considering pulmonary artery end-diastolic pressure. While patients with a stroke work index of more than 60 g m/m^2 had a very good prognosis (with pulmonary artery end-diastolic pressure values below 18 mmHg nobody died), patients with stroke work index values below 20 g m/m^2, irrespective of
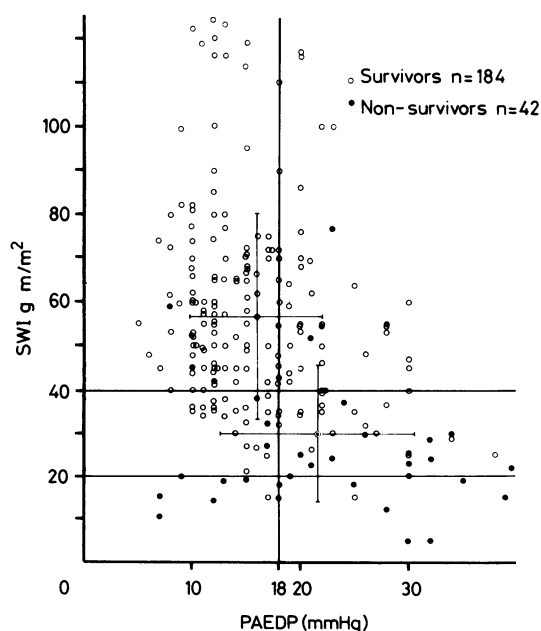

Fig. 2 *Distribution of survivors and non-survivors among 226 patients with acute myocardial infarction according to the initial values of stroke work index (SWI) and pulmonary arterial end-diastolic pressure (PAEDP). (See Table 3.)*

Table 3   *Relation between initial values of stroke work index (SWI) and pulmonary arterial end-diastolic pressure (PAEDP) and mortality in 226 patients with acute myocardial infarction*

Value of SWI (g m/m²)	Total number of patients					Patients with PAEDP <18 mmHg					Patients with PAEDP ≥ mmHg				
	No.	Survivors No.	%	Non-survivors No.	%	No.	Survivors No.	%	Non-survivors No.	%	No.	Survivors No.	%	Non-survivors No.	%
>60	73	72	98·6	1	1·4	53	53	100	—	—	20	19	95	1	5·0
41–60	77	68	88·3	9	11·7	51	47	92·2	4	7·8	26	21	80·8	5	19·2
21–40	58	42	72·4	16	27·6	27	24	88·9	3	11·1	31	18	58·1	13	41·9
≤20	18	2	11·1	16	88·9	7	1	14·3	6	85·7	11	1	9·1	10	90·9
Total	226	184	81·4	42	18·6	138	125	90·6	13	9·4	88	59	67·0	29	33·0

pulmonary artery end-diastolic pressure, had a poor prognosis (mortality rate 90%). Between 20 and 40 g m/m² there was a certain grey zone with mortality rates between 11·1 and 41·9%. But, again, with pulmonary artery end-diastolic pressure values below 18 mmHg, the prognosis was better. The mean value of stroke work index in survivors was $57·4 \pm 23·7$ g/m² and in non-survivors $30·2 \pm 16·4$ g/m².

Of the 42 non-survivors, 41 died from cardiac causes and one from apoplexy (Fig. 3, Table 4). Cardiogenic shock occurred in 24 patients,
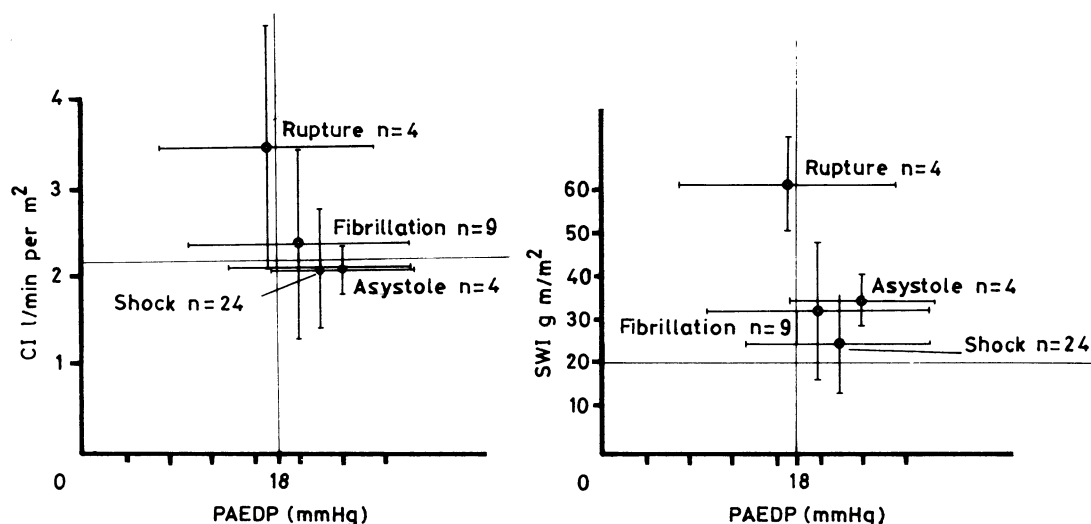

Fig. 3   *Initial findings of cardiac index (CI) (on left) and stroke work index (SWI) (on right) vs. pulmonary arterial end-diastolic pressure (PAEDP) in 41 non-survivors in relation to cause of death. (See Table 4.)*

Table 4   *Initial haemodynamic findings in non-surviving patients: separation according to cause of death*

Haemodynamic variable	(1) Cardiac rupture (No.=4)	(2) Ventricular fibrillation (No.=9)	(3) Terminal asystole (No.=4)	(4) Cardiogenic shock (No.=24)	(5) Fibrillation, asystole, and shock (No.=37)	Significance (1) vs (5)
CI (1/min × m²)	3·5±1·4	2·4±1·1	2·1±0·3	2·1±0·7	2·2±0·8	p<0·005
PAEDP (mmHg)	17·3±9·8	20·1±10·5	24·0±6·7	22·6±8·7	22·2±8·8	NS
SWI (g m/m²)	61·0±11·0	31·7±16·3	34·0±5·9	22·9±11·7	26·2±13·1	p<0·0001

Note: The figures for cardiac index (CI), pulmonary arterial end-diastolic pressure (PAEDP), and stroke work index (SWI) are group means ± standard deviation.

irreversible ventricular fibrillation in nine, terminal asystole in four, and cardiac rupture in four. The initial haemodynamic values of cardiac index, pulmonary artery end-diastolic pressure, and stroke work index were similar in patients who died from cardiogenic shock, from fibrillation, and from asystole. In those who died from cardiac rupture, however, cardiac and stroke work index were significantly higher and pulmonary artery end-diastolic pressure was lower compared with the other groups. Classifying the haemodynamic findings in relation to the time elapsed since the onset of infarction (Fig. 4, Table 5) within the first four hours (time group A) we found relatively high values of cardiac and stroke work index in survivors, while these values were already lowered in non-survivors. The average pulmonary artery end-diastolic pressure in survivors was at the upper limit of normal and was slightly raised in non-survivors. From the fifth hour on (time groups B and C) cardiac and stroke work indices in survivors

and in non-survivors were lower, though not significantly so. Among patients admitted 13 to 24 hours after onset of infarction (time group D) who died in hospital, cardiac and stroke work indices were higher than in patients admitted earlier. The pulmonary artery end-diastolic pressure in this subgroup of non-survivors was also raised. Most of the 18 patients admitted more than 24 hours after onset (time group E) had normal cardiac index, stroke work index, and pulmonary artery end-diastolic pressure; the two non-survivors in this group presented the worst findings of the group.

The mortality rate in the group presenting earliest (group A) was relatively high at 19%. The rate fell to 11% and 15% in the groups presenting later (groups B and C). In patients of group D, who were admitted between 13 and 24 hours after infarction, mortality was again high at 36%. Only two of 18 patients in group E died. In all five time groups the initial pulmonary artery end-diastolic pressure of the
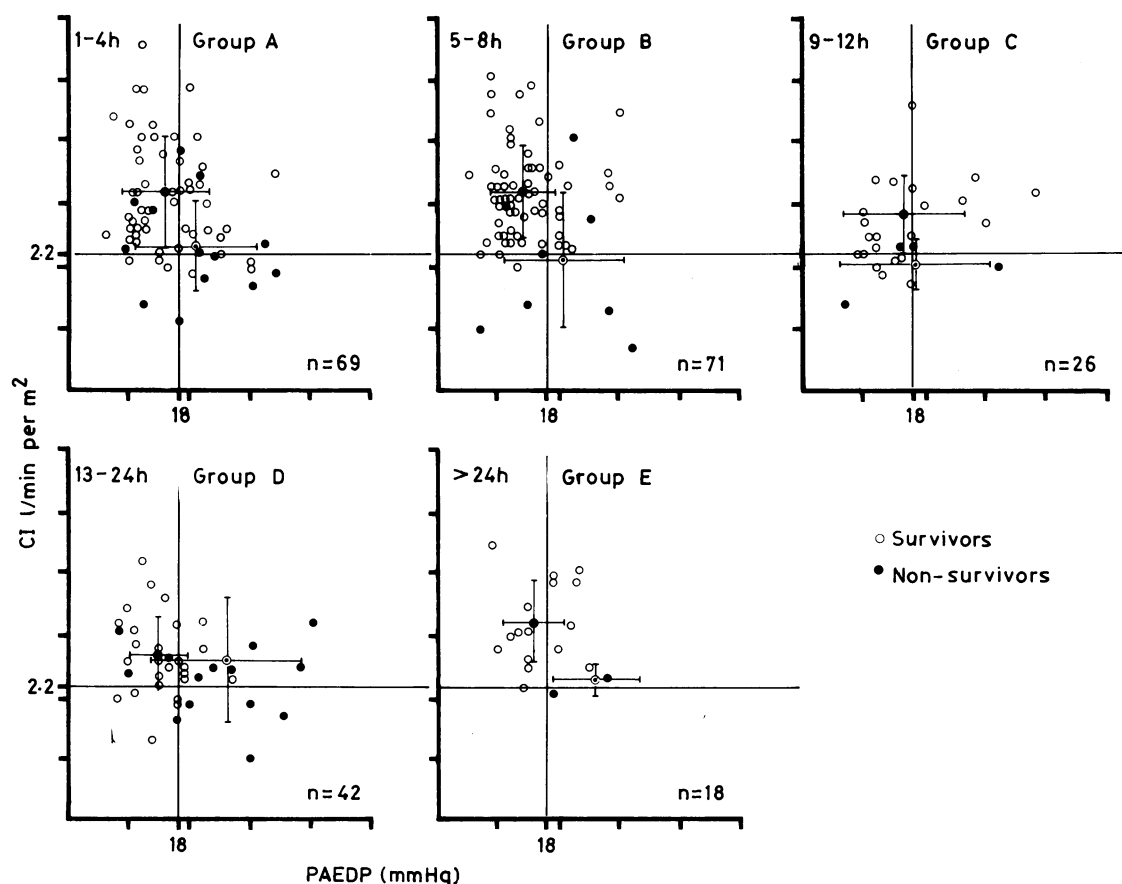

Fig. 4 *Initial haemodynamic findings of cardiac index (CI) and pulmonary arterial end-diastolic pressure (PAEDP) in relation to different admission times after onset of infarction. Mean values and standard deviation of survivors and non-survivors. (See Table 5.)*

Table 5 *Initial measurements of cardiac index (CI), stroke work index (SWI), and end-diastolic pulmonary artery pressure (PAEDP) in relation to time elapsed after onset of infarction*

Time after onset of infarction	Survivors (No. = 184)			Non-survivors (No. = 42)			Mortality (18·6%)
	CI	SWI	PAEDP	CI	SWI	PAEDP	
1–4 h (Group A)	3·2±0·9	58·8±28·5	16·1±6·3	2·3±0·8 (p<0·002)	29·9±14·9 (p<0·001)	20·2±8·2 (p$\frac{3}{4}$0·056)	13/69 (19%)
5–8 h (Group B)	3·2±0·8	63·2±24·7	15·2±5·4	2·1±1·2 (p<0·0015)	21·9±13·8 (p<0·0001)	19·8±8·5 (p<0·04)	8/71 (11%)
9–12 h (Group C)	2·8±0·7	55·1±24·3	16·9±7·7	2·0±0·4 (p<0·04)	24·8±10·4 (p<0·03)	18·3±10·3 (NS)	4/26 (15%)
13–24 h (Group D)	2·7±0·6	52·0±17·6	15·3±4·5	2·6±1·0 (NS)	35·3±18·7 (p<0·008)	24·1±9·5 (p<0·0003)	15/42 (36%)
>24 h (Group E)	3·2±0·7	60·6±18·2	15·9±4·4	2·3±0·2	37·5±24·8	23·5±6·4	2/18 (11%)

CI = l/min × m²; SWI = g m/m²; PAEDP = mmHg (p values between survivors and non-survivors).
Note: Results are group means ± standard deviation.

survivors was almost identical, with a value of 16 mmHg. In the group of non-survivors the pulmonary artery end-diastolic pressure was higher and varied in the different time groups. In survivors significant differences in cardiac index were found only between groups B and C (p<0·05) and between groups D and E (p<0·02).

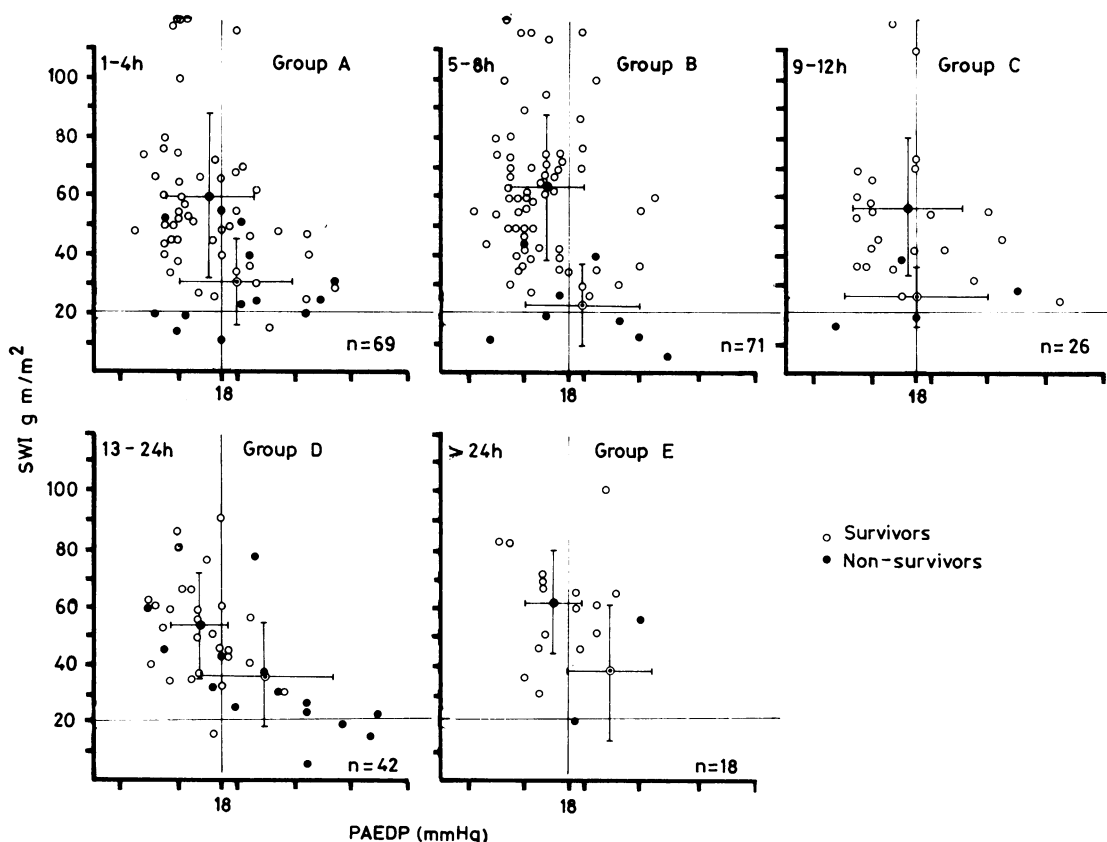The majority of patients dying later of cardiogenic

Fig. 5 *Initial haemodynamic findings of stroke work index (SWI) and pulmonary arterial end-diastolic pressure (PAEDP) in relation to different admission times after onset of infarction. Mean values and standard deviation of survivors and non-survivors. (See Table 5.)*

Table 6 *Cause and day of death after onset of acute myocardial infarction in 42 patients subdivided into groups A to E according to time between onset of infarction and admission to hospital*

Time group	Cause of death	No.	Day of death
A	Shock	10	1, 1, 1, 2, 2, 3, 5, 5, 7, 9 (3·6±2·8)
1–4 h	Fibrillation	1	5
	Asystole	1	2
	Rupture	1	3
B	Shock	5	1, 1, 1, 5, 7 (3·0±2·8)
5–8 h	Fibrillation	3	1, 3, 3 (2·3±1·2)
C	Shock	1	1
9–12 h	Fibrillation	1	13
	Asystole	2	1, 1
D	Shock	8	1, 1, 2, 2, 2, 3, 7, 29 (5·±9·5)
13–24 h	Fibrillation	3	2, 2, 5 (3·0±1·7)
	Asystole	1	13
	Rupture	2	11, 20
	Apoplexy	1	33
E	Fibrillation	1	4
>24 h	Rupture	1	4

Note: Means ± standard deviation for day of death are given in parenthesis.

shock (Table 6) were admitted either within the first eight hours or were delayed between 13 and 24 hours after onset of infarction. Like those dying from intractable ventricular fibrillation and terminal asystole, they died mostly within the first three days. The patients with cardiac rupture died three, four, 11, and 20 days after infarction; three of them were admitted more than 12 hours after the onset of symptoms.

Discussion

According to Killip and Kimball,[15] the clinical staging of cardiac failure in acute myocardial infarction is only reliable in large infarctions with manifest left ventricular failure. In smaller infarctions the clinical signs of pump failure may follow raised pulmonary artery end-diastolic pressure with a delay of up to 12 hours.[6 8 10 16] On the other hand, râles may sometimes still be heard despite return to normal of left ventricular filling pressure. In comparison with purely clinical examination pulmonary arterial pressure measurements via balloon catheter[1–7] allow an immediate, quantitative evaluation of the haemodynamic disturbances and of the effects of treatment.[16–20] Thus pure clinical, non-invasive techniques are not reliable enough to form the basis for prognosis and evaluation of treatment. Though the haemodynamic findings at any one moment in the individual patient may be useful, they must be regarded critically as isolated values. They ought to form part of a longitudinal analysis and be correlated with the whole clinical picture. They are ideally supplemented by two-dimensional echocardiography

[21 22] and scintigraphy.[23] The calculation of infarct size from the creatinine kinase washout curve[5 24] in the individual case has not proved sufficiently reliable.[25]

When considering the significance of haemodynamic findings in the early phase of acute myocardial infarction one has to consider the augmentation of stiffness of the infarcted myocardial area by oedema and cellular infiltration which occurs within the first few hours after coronary occlusion. Because of these changes in compliance, left ventricular filling pressure, as reflected by pulmonary artery end-diastolic pressure, rises. Subsequently, the stiffness resolves and pulmonary artery end-diastolic pressure accordingly falls.[2 7 17 18 26 27] The increased secretion of endogenous catecholamines in this early phase increases simultaneously the contractility of the residual myocardium, causing cardiac and stroke work index to rise. Alterations in left ventricular filling pressure, which occur in most cases of transmural infarction, are not primarily indicative of the prognosis. As long as cardiac and stroke work indices remain normal, pulmonary artery end-diastolic pressure values between 18 and 22 mmHg in this early phase of the infarction are not indications for the immediate use of vasodilators, but should encourage continuous close observation. If cardiac and stroke work index values fall, treatment with positive inotropic agents or even intra-aortic balloon pulsation have to be considered.[2 16 18] Despite this haemodynamic instability in the acute phase, consideration of the initial cardiac index, stroke work index, and pulmonary artery end-diastolic pressure gives some prognostic information.[3 4 6 7 9 28] With certain reservations, prognostications for the year after the infarction may also be made from them.[19 28 29] In our series 13 out of 23 patients with pulmonary artery end-diastolic pressure values greater than 28 mmHg and seven out of nine patients with values greater than 32 mmHg died; this corresponds with other reported experience.[6 9 10 12 16] With initial cardiac index values below 2·2 l/min per m^2 20 out of 44 patients died, and below 1·8 l/min per m^2, 12 out of 15. None of the eight patients with cardiac index less than 1·4 l/min per m^2 survived. When the stroke work index was below 40 g m/m^2 32 out of 76 patients died, and 16 out of 18 patients with a stroke work index of less than 20 g m/m^2 succumbed; again, similar reports can be cited.[4 7 8 10 12 19] Seventy of the 73 patients with values over 60 g m/m^2 survived to leave hospital. One patient died from cardiac rupture 20 days after the onset of infarction.

The combined analysis of cardiac index and pulmonary artery end-diastolic pressure is superior to consideration of the single items.[2–7 16–20] Consideration of pulmonary artery end-diastolic pressure

alone, without reference to some indication of the degree of pump failure in terms of cardiac and stroke work index, can be misleading. If the cardiac index is below 2·2 l/min per m^2 and the pulmonary artery end-diastolic pressure at the same time exceeds 18 mmHg, mortality is approximately 70%. The analysis of stroke work index shows a certain grey zone between 20 and 40 g m/m^2, where the level of pulmonary artery end-diastolic pressure is crucial. In our series with pulmonary artery end-diastolic pressure values below 18 mmHg mortality was 11%; with values above 18 mmHg it rose to 42%. With stroke work index values of less than 20 g m/m^2 mortality was 91% and pulmonary artery end-diastolic pressure values did not provide any additional prognostic information. The seven patients in this group who had pulmonary artery end-diastolic pressure below 18 mmHg had predominant right ventricular infarction and the high mortality in this subgroup (86%) perhaps reflects the poor adaptability and limited contractile reserve of the right ventricle.

Patients with a hyperkinetic left ventricle – cardiac index over 3·5 l/min per m^2, stroke work index over 60 g m/m^2, and pulmonary artery end-diastolic pressure below 18 mmHg – generally had a good prognosis, but all four patients with cardiac rupture had remarkably high cardiac index and stroke work index findings. Because of raised catecholamine stimulation the contractility of the non-ischaemic myocardium is greatly increased. Thus the infarcted area tends to be expanded in systole and left ventricular wall stress is enhanced.[2] This increase of wall tension can lead to an expansion of the infarcted area and finally to cardiac rupture.[30] This mechanism may occur especially in patients like ours who are slow in reaching hospital. In this hyperkinetic state the early administration of beta-blockers may prevent the expansion of the infarction and subsequent myocardial rupture.

There is evidence that the final outcome, including mortality, in acute myocardial infarction depends on the time between onset of infarction and the introduction of relevant treatment after hospital admission.[7] [12] Our patients admitted within the first four hours often had large infarctions and had perhaps been sent to hospital at an early stage because of severe symptoms. Some were in shock on arrival and responded poorly to treatment. Thus, the group mortality of about 20% was quite high. Those patients admitted five to 12 hours after onset had a group mortality of 12%; their infarctions were mostly smaller, and the clinical symptoms, the degree of pain, and the signs of cardiac failure less impressive. Sometimes, the admission of patients even with larger infarctions was delayed to the 13th or 14th hour. The detailed questionnaire disclosed that this delay was partly caused by the patients or their relatives and partly by the family physicians. Several of them had already developed pump failure with pulmonary congestion, acidosis, and ventricular rhythm disturbances at the time of admission. The presence of several such high-risk patients within the 13 to 24 hour admission group contributed to its high mortality figure of 36%. Most patients in the small group admitted 24 hours or more after the onset of symptoms had relatively small infarctions with stable haemodynamics and a good prognosis (mortality 11%).

If haemodynamic measurements in the acute phase of the myocardial infarction are considered without reference to the age of the infarction, findings in patients who have just developed features of shock are mixed up with those in haemodynamically compensated patients with small infarctions in a later phase of the disease. The large American multicentre study[10] defines an interval of 36 hours after onset of infarction as the criterion of inclusion. This large time interval brings together a broad spectrum of patients at different stages of the acute disease. A pulmonary artery end-diastolic pressure value of 22 mmHg found within the first 12 hours of an infarction may be solely the result of initial left ventricular stiffness and become normal within the next few hours without any treatment and with a good prognosis. If such a value is registered during the 24th hour it may be a sign of an expanding infarction with incipient pump-failure. It could, however, be a good intermediate finding in a situation improving after initial pump failure, with even higher pulmonary artery end-diastolic pressure values. Like Shell et al.,[14] we found significant prognostic information from the longitudinal comparison of pulmonary artery end-diastolic pressure and cardiac and stroke work indices within the first 48 to 72 hours.[12] If pulmonary artery end-diastolic pr
18 mmHg (mortali
2·2 l/min per m^2
25 g m/m^2 (mortality 5% and 4%, respectively), the prognosis of these small, monophasic infarctions was good. If initially abnormal pulmonary artery end-diastolic pressure and cardiac and stroke work indices became normal within the first two days, the outcome was still not too bad (mortality 10%, 19%, and 25%). If, however, pulmonary artery end-diastolic pressure rose during the first three days (mortality 23%) or cardiac and stroke work indices fell, the prognosis was considerably worse (mortality 58% and 40%). Some of these patients were experiencing recurrent ischaemia and perhaps a biphasic pattern of infarction, with or without infarct expansion. With persistently raised pulmonary artery end-diastolic pressure and subnormal cardiac and stroke work indices (mortality

47%, 65%, and 88%) the prognosis was gloomy. In these large infarctions with severe myocardial damage the residual areas of the ventricle were usually unable to maintain adequate cardiac pumping function.

All survivors, irrespective of the timing of their first haemodynamic measurements, had surprisingly similar values of pulmonary artery end-diastolic pressure at around 16 mmHg. As a rule, non-survivors admitted after the 13th hour had raised pulmonary artery end-diastolic pressures. Cardiac and stroke work indices within the first eight hours were usually somewhat raised and fell later. The capacity of the infarcted ventricle temporarily to pump more blood may be partly an effect of the raised left ventricular filling pressure and partly an effect of increased catecholamine drive. In large infarctions with destruction of much of the left ventricle and in predominantly right ventricular infarctions these compensatory mechanisms are ineffective and pump failure supervenes.

The most unstable phase in the acute myocardial infarction is the first 24 hours. Changes may occur quickly and without warning. If the haemodynamic findings remain stable for 12 to 15 hours during this first day, however, we feel that haemodynamic monitoring can be discontinued. Beyond this time we found the chance of deterioration in previously stable patients to be below 10%.[12] Late deterioration is mostly accompanied by the new onset of pain, or by electrocardiographic or creatinine kinase changes. In such circumstances the prognosis is worse and it is our belief that haemodynamic monitoring should be recommended, particularly to enable treatment to be chosen and adjusted optimally.

The patients included in this paper were all treated in the classical way with vasodilators, positive inotropic agents, beta-blockers, and fluid.[2 16 18] The haemodynamic measurements and follow-up studies will form the basis for comparison of findings in those patients who are now treated in our hospital with selective intracoronary thrombolysis and, in some suitable cases, also with the adjacent application of transluminal balloon dilatation of the underlying coronary artery stenosis. This comparison will disclose whether this aggressive revascularisation within the first eight hours after the onset of the infarction is more capable than conventional treatment of saving myocardium, of preventing an enlargement or a relapse of ischaemia, and of improving prognosis.

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